Superior Mesenteric Artery Syndrome in a 20-year-old Athletic Female with Abdominal Pain

Ashley Mingshin Lee^{1,2}, Harrison Xiao Bai², Ying-Jie Zou¹, Zhi-Li Wang¹, Dong-Xu Qiu¹, Hai-Yun Tang¹, Li Yang¹

¹Department of Neurology, Second Xiangya Hospital, Central South University, Changsha, Hunan 410011, China ²Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania, USA

To the Editor: A 20-year-old female college student and soccer player presented to the Emergency Department with acute onset of abdominal pain, nausea, and vomiting. On the morning of presentation, she woke up with sudden onset of epigastric pain that was constant and 10/10 in severity. The patient denied any obstipation, constipation, diarrhea, any recent trauma to the abdomen, or any potential inciting event. Of note, the patient did endorse previous episodes of postprandial epigastric pain, but it was never this severe. Her past medical and surgical history was unremarkable.

Physical examination revealed an athletic female (height: 1.65 m, weight 45.0 kg, body mass index: 16.5 kg/m²) who appeared uncomfortable on the stretcher. Her vitals were stable. Her abdomen was soft and non-distended. There was moderate epigastric tenderness without guarding or rebound tenderness. A computed tomographic (CT) scan of the abdomen revealed moderate dilatation of the duodenum that measured up to 3.4 cm with an abrupt caliber change [Figure 1a and b] and decreased angle between the aorta and superior mesenteric artery (SMA) [Figure 1c]. The patient was diagnosed with SMA syndrome.

SMA syndrome describes a constellation of symptoms due to duodenal compression by the SMA and abdominal aorta. In normal anatomy, the third portion of duodenum courses posteriorly to the SMA and anteriorly to the abdominal aorta at the level of the third lumbar vertebrae (L3). These three structures, along with the left renal vein, are surrounded by mesenteric fat pad and lymphatics. In patients with SMA syndrome, the aorto-mesenteric angle is decreased to less than 25° (normal: 38–65°) and distance less than 8 mm (normal: 10–28 mm). [1,2] SMA syndrome may be associated with entrapped left renal vein, or nutcracker, syndrome. Common etiologies of SMA syndrome include attenuation of mesenteric fat secondary to weight loss (most common) or surgical alteration of anatomy.

Patient's presentation, physical exam, and laboratory findings are consistent with small bowel obstruction but are otherwise nonspecific. The patient's symptoms may include nausea, vomiting, and epigastric tenderness in either acute or chronic settings. The patient may endorse recent weight loss or abdominal surgery. Lying in positions that relieve tension between the SMA and aorta space such as the left decubitus or knee-to-chest may help alleviate symptoms.

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Differential diagnosis of SMA syndrome includes other causes of acute abdominal pain such as other causes of bowel obstruction, mesenteric ischemia, and gastroesophageal reflux disease (GERD). It may be difficult to differentiate SMA syndrome from other causes of bowel obstruction as both of them have similar presentations and laboratory findings. The patients with other causes of small bowel obstruction tend to have more comorbidities such as history of neoplasm, prior irradiation, and intestinal inflammation such as Crohn's disease. Prior abdominal surgery is a risk factor for both SMA syndrome and other causes of bowel obstruction. Mesenteric ischemia is most commonly caused by atherosclerotic or embolic events in the mesenteric vasculature. Therefore, patients with mesenteric ischemia are likely to have significant cardiovascular risk factors such as advanced age, diabetes mellitus, and coronary artery disease, etc. Approximately half of the patients have abdominal bruits on physical exam. The patient typically presents with recurrent epigastric pain that occurs within the first hour of eating. GERD is a diagnosis of exclusion in the Emergency Department. The patient's symptoms are nonspecific, and laboratory and radiologic studies are without significant derailment.

Definitive diagnosis of SMA syndrome is reached upon direct visualization of the decreased aorto-mesenteric angle (<25°) and/or distance (<8 mm) on CT, magnetic resonance, ultrasonography, [3] or conventional angiography. Additional diagnostic imaging criteria include duodenal obstruction with abrupt cut-off in the third portion in setting of active peristalsis, and/or anatomic abnormalities such as high fixation of duodenum by the ligament of Treitz and anomaly of SMA. Plain films and upper gastrointestinal series may demonstrate dilated proximal duodenum and other findings suggestive of small bowel obstruction.

SMA syndrome can be managed conservatively with small bowel decompression and electrolyte correction. In patients with weight

Address for correspondence: Dr. Li Yang, Department of Neurology, Second Xiangya Hospital, Central South University, Changsha, Hunan 410011, China E-Mail: yangli762@gmail.com

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Figure 1: Abdominal computed tomographic image with oral and intravenous contrast demonstrating a dilated duodenum to 3.4 cm (a) with an abrupt change of caliber (b) on axial images (black arrow) and a narrowed angle between the superior mesenteric artery and abdominal aorta (black lines with angle of 20°) on sagittal image (c).

loss as the underlying etiology, nutrition therapy with psychiatric counseling offers a conservative treatment option with high success rate. In patients who are refractory to nutrition therapy or have anatomic abnormalities as the underlying etiology, surgical intervention such as Strong's procedure, [4] gastrojejunostomy, or duodenojejunostomy^[5] may be considered. However, the long-term outcomes for such surgical techniques have not been thoroughly evaluated.

Our patient was managed conservatively and discharged with outpatient follow-up. She was encouraged to increase her daily caloric intake. At 2-month follow-up, she had gained 5 kg and had remained asymptomatic.

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Conflicts of interest

There are no conflicts of interest.

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